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# Comment on: IgA nephropathy with early kidney disease is associated with increased arterial stiffness and renin-angiotensin system activity

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**Abstract**

We read with great interest the article by Abdi-Ali et al. entitled "IgA nephropathy with early kidney disease is associated with increased arterial stiffness and renin–angiotensin system activity" in the recent issue of *Journal of the Renin–Angiotensin–Aldosterone System*. In a study on 10 normotensive IgA nephropathy (IgAN) subjects with early kidney disease and 10 gender- and blood pressure-matched healthy controls, blood pressure and arterial stiffness, expressed as pulse wave velocity and aortic augmentation index, at baseline and in response to 60 min of angiotensin II (AngII) infusion were measured. They found that IgAN patients had an increased aortic augmentation index and a trend towards increased circulating renin–angiotensin system components at baseline, compared with controls.

**Keywords**

IgA nephropathy, vasculopathy, angiotensin II

## Dear Editor-in-Chief,

We read with great interest the article by Abdi-Ali et al. entitled "IgA nephropathy with early kidney disease is associated with increased arterial stiffness and renin–angiotensin system activity" in the recent issue of *Journal of the Renin–Angiotensin–Aldosterone System*.[1] In a study on 10 normotensive IgA nephropathy (IgAN) subjects with early kidney disease and 10 gender- and blood pressure-matched healthy controls, blood pressure and arterial stiffness, expressed as pulse wave velocity and aortic augmentation index, at baseline and in response to 60 min of angiotensin II (AngII) infusion were measured. They found that IgAN patients had an increased aortic augmentation index and a trend towards increased circulating renin–angiotensin system (RAS) components at baseline, compared with controls.[1] They also detected that IgAN was associated with reduced arterial sensitivity to angiotensin II (AngII) challenge. They concluded that IgAN is associated with increased arterial stiffness and decreased AngII responsiveness, a marker of increased RAS activity.[1]

We would like make some comments related to the vascular pathology in IgAN. To evaluate the role of vascular disease in IgAN patients, we recently conducted a study on 136 IgAN patients. We found a significant correlation between the scores of arteriolosclerosis and serum creatinine. This correlation for the intensity of intimal fibrosis of interlobular artery was also significantly positive. The correlation of arteriolosclerosis with proteinuria was also significantly positive. We concluded that the association of vasculopathy with serum creatinine and amount of proteinuria further reinforces the role of vasculopathy in the aggravation of IgAN.[2] There are very few studies that have reported to date on the significance of vasculopathy or its etiology in IgAN. It is, however, clear that the vascular disorders in IgAN are accompanied by aggravation of renal failure and increased cardiovascular risk.[3–5] However, the pathogenesis of the vascular disorders is still not completely understood. The study by Abdi-Ali et al.[1] clearly shows the role of AngII in the aggravation of vasculopathy of IgAN.

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Previously, El Karoui et al. showed that 53% of 128 IgAN patients had the morphologic lesions of thrombotic microangiopathy (TMA).[6] They concluded that morphologic lesions of TMA are frequent in IgAN.[6] Interestingly, it has been speculated that AngII takes part in the initiation or progression of TMA.[7–12] The findings of our study[2] and the study conducted by El Karoui et al.[6] further support the results of the present study. However, vasculopathy in IgAN requires more investigation in larger studies.

## Authors' contributions

MM and HN wrote the paper equally.

## Conflict of interest

None declared.

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